Relating biological response modeling to air quality criteria

RELATING BIOLOGICAL RESPONSE MODELING TO AIR QUALITY CRITERIA

by: A. Venkatram

Ontario Ministry of the Environment
Air Resources Branch
Air Quality and Meteorology Section

JANUARY, 1978

ABSTRACT

A method is proposed to relate simple biological response modeling to air quality criteria. We also suggest a procedure using the biological response curve in conjunction with measured pollutant concentrations for setting emission levels to ensure compliance with air quality standards. A byproduct of our analysis is an air pollution index which avoids the arbitrariness of commonly used indices.

1. Introduction

In a series of papers (see Larsen et al., 1977) Larsen has proposed a rational procedure to set air quality criteria. His method is based on an extensive study of the response of vegetation and mice to exposure from various pollutants. Larsen found that the pollutant concentration required to produce a specified effect varied as some power of exposure time. In other words, the concentration versus exposure duration plotted as a straight line on a log-log graph. Clearly this type of plot is useful for the derivation of air quality criteria (Munn et al., 1977). Instead of concentrations, (which are often chosen independently of each other) at distinct averaging times we have an "operating" curve of air quality criteria as a continuous function of averaging time. While this approach is preferable to commonly used criteria setting procedures it is still a static method in that it does not account for the continuous response of organisms to pollutant doses. This disadvantage becomes clear when we consider a situation where say two hours of concentrations slightly below the criterion are followed by one hour in which the criterion is violated. Obviously, this one hour violation has a greater effect than that preceded by hours of zero concentration. Air quality criteria do not distinguish between these two situations.

In this paper we present a simple dynamic model of biological response to pollution. We show that this type of model, which has been used by Saltzman (1970) to study biological transfer functions, can also be utilized to derive air quality criteria.

2. Model

The simplest model of biological response can be represented by the equation

$$\frac{\partial E}{\partial t} = \beta C_a - \alpha E \qquad (1)$$

In (1) α and β are proportionality constants, and E represents the effect produced by the ambient pollutant concentration C_a. It is clear that we assume that the pollutant uptake rate is proportional to C_a and the detoxification rate is proportional to E. The meaning of (1) is clarified if we take C_a to be the concentration of CO and E to be that of carboxyhemoglobin (COHb). There is some research (Roach,1966) which indicates that the response of COHb in human blood to CO exposure is modeled fairly well by the solution of (1). Roach also shows that a wide variety of pollutants elicit this type of response from critical organs in the human body. We feel that the extension of the model to vegetation response is not unreasonable.

For a constant C_a, the solution of (1) is

$$E(t) = E(0)\exp(-\alpha t) + \frac{\beta C_a}{\alpha}\left[1 - \exp(-\alpha t)\right] \qquad (2)$$

We assume that a specified degree of pollutant effect corresponds to some definite level of E. Then, taking E(o) = O, the concentration required to produce a specified effect over a given exposure time t is given by

$$C_{at} = \frac{C_m}{(1-\exp(-\alpha t))} \qquad (3)$$

In (3), C_m is the pollutant concentration which produces the specified effect when $t \to \infty$. In other words, C_m is the concentration to which the organism can be exposed continuously with no "adverse" effects. It is noted that C_m essentially specifies the degree of injury. As β is arbitrary, we take $\alpha = \beta$ so that the allowable effect becomes C_m, the long term concentration. For small t, Eq. 3 reduces to

$$\alpha C_{at} t = C_m \qquad (4a)$$

This shows that equal doses produce equal effects only at small exposure times. This can be clarified by rewriting (3) as

$$C_m = \frac{D\,(1 - \exp(-\alpha t))}{t} \qquad (4a)$$

As C_m is the effect variable, the effect is proportional to the dose $D = C_{at}\, t$ only for small t. In fact, for a constant dose the effect decreases as the exposure time increases. This result is compatible with experimental findings (Heck and Brandt, 1977).

3. Air Quality Criteria

It is clear that Eq.(3) can be used to derive air quality criteria. In Fig.1 we have plotted (3) on a log-log graph. The curve, which we refer to as the biological response curve BR, is specified by α (which is the inverse of the biological response time τ_b) and the effect C_m. In the figure, the straight line D_1 is a representation of measured maximum concentrations as a function of averaging time. The form of D_1 is based on several studies in urban areas (Larsen, 1971) which indicate that concentration versus averaging time plots as a straight line on a log-log graph. If we desire to prevent biological effects above a level characterized by C_m it is clear that measured concentrations given by D_1 have to be reduced. Between the averaging times t_A and t_B expected maximum concentrations are above the limits set by BR. In order to prevent pollutant induced injury we have to shift the curve D_1 so that it lies below BR at all averaging times. The line D_2, which is tangential to BR at C, satisfies the air quality criteria set by BR. It is clear from the figure that the critical averaging time t_c and the associated concentration is determined by the condition

Slope of BR at t_c = Slope of D_1 (= slope of D_2)

We note that emission reduction does not change the slope of the measured concentration. It is readily seen that the above condition reduces to

$$x \exp(-x)/(1-\exp(-x)) = p \qquad (5)$$

where $x = \alpha t$ and p is the negative of the slope of D_1. We note that (5) is independent of C_m. Thus, once we know x as a function of p, we can determine the point through which D_1 should pass by specifying the effect C_m.

Let us illustrate the procedure described in the previous paragraph by considering carbon monoxide, a gas whose effects on the human body are fairly well known. It is recalled that CO standards are based on criteria relevant to the human body. Calculations by Roach (1966) indicate that τ_b for CO is 2.88 hr. Then, assuming that the 1-hour (MOE,Canada) standard for CO of 30 ppm is related to a permissible level of COHb, we can compute C_m which turns out to be 8.80 ppm. With the biological response curve completely specified we can compute t_c and the associated CO concentration for a specific urban area. For a typical value of p we choose 0.15 from a study by McGuire and Noll(1971). From Fig.2 we find that for p = 0.15, x is 3.06,a value which translates into t_c= 8.81 hr. From (3) we can compute the concentration as 9.23 ppm. The calculation indicates that for an area with p = 0.15 we can satisfy the criteria set by the BR curve for CO by ensuring that the maximum concentration for an averaging time of 8.81 hr. is less than or equal to 9.23 ppm. It is worthwhile to clarify the meaning of this criterion. What we are saying is that by meeting the 8.81 hr. concentration requirement we do not have to worry about concentrations at other averaging times. This would be true, of course, only if the maximum concentrations did not deviate from the D curve. As p is a function of time and area for the same pollutant (McGuire and Noll,1971), it is clearly not very meaningful to derive criteria and hence standards from the D line. The criteria are set by the BR curve. While the D line can be used to help us to meet the requirements of the BR curve, it cannot specify a set of objective standards.

The discussion of the previous paragraphs shows that the concept of the biological response curve can be very useful in the establishing of air quality criteria. However, its utilization forces us to replace time varying concentrations by averages. Clearly, the response to a varying concentration over a specified interval of time is not equivalent to that

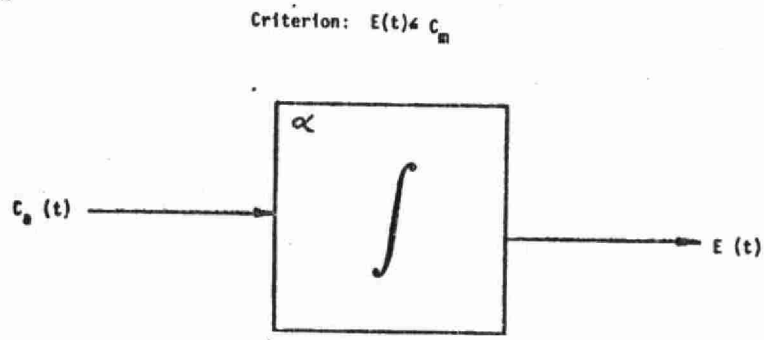
to a constant average concentration over the same time period. In the next section, we propose a dynamic model of biological response in order to improve upon the "static" nature of the derivation of criteria we have just discussed.

4. Dynamic Model and An Air Pollution Index

The general solution of Eq.(1) can be written as

$$E(t) = E(0)\exp(-\alpha t) + \alpha \int_0^t C_a(\tau)\exp(\alpha\tau)\exp(-\alpha t)\, d\tau \quad (6)$$

As $C_a(t)$ is known, it is a simple matter to compute $E(t)$. Then, from the previous discussion it is clear that the statement of our "dynamic" criterion is that $E(t)$ should not exceed C_m which specifies the level of allowable injury. This concept can be expressed in a diagram.



What we are essentially doing is transforming the ambient concentration $C_a(t)$ to $E(t)$ through the parameter α ($= 1/\tau_b$). The integral sign indicates that $E(t)$ represents the cumulative effect due to $C_a(t)$.

The preceding discussion suggests that we can construct an air pollution index by normalizing Eq.(6) with C_m. Then the equation describing the Index $I = E(t)/C_m$ is:

$$I(t) = I(0)\exp(-\alpha t) + \frac{\alpha}{C_m}\int_0^t C_a(\tau)\exp(\alpha\tau)\exp(-\alpha t)\, d\tau \quad (7)$$

We notice that I ranges from 0 to 1 when the cumulative effect of pollutants is within allowable limits. An index greater than one indicates a problem.

An approximate form of (7) can be written as

$$I(t+\Delta t) = I(t)\exp(-\alpha\Delta t) + \frac{\bar{C}_a(t+\Delta t)}{C_m}\left[1-\exp(-\alpha\Delta t)\right]$$

$$\bar{C}_a(t+\Delta t) = \frac{1}{\Delta t}\int_t^{t+\Delta t} C_a(\tau)\,d\tau \qquad (8)$$

The attractive feature of the index in (8) is that it accounts for the cumulative effect of pollutants. Furthermore, as (8) mimicks the actual biological response, I is a measure of the injury caused by the pollutant in question. Clearly, this is a vast improvement over the commonly available air pollution indices (Thom and Ott,1975) the basis of which are admittedly arbitrary.

Knowledge of C_m and α for commonly found pollutants will allow us to compute indices for each of the pollutants. For the purpose of reporting to the public, individual index values or the maximum value can be used. It is noted that the suggested index does not account for synergistic effects. But then, as far as we are aware, no other index does so in a scientifically acceptable manner.

5. Determination of α and C_m

As our model of biological response depends on the two parameters α and C_m, in theory, we can specify BR by experimentally determining two points on the curve. Suggestions on the details of an experimental program to determine these parameters are outside the scope of this paper. However, it is worthwhile to deduce α and C_m from secondary air quality standards, assuming of course, that they are "based" on points on the BR curve.

One of the graphs in Fig.3 is a BR curve for vegetation based on U.S.-E.P.A. secondary standards (3-Hr., 24-Hr., and annual) for SO_2. By choosing two standards at a time we can fit curves to generate three sets of C_m and α ($= 1/\tau_b$). At the present time there is not enough information to choose between the BR curves. However, as the 3-Hr. and 24-Hr. concentrations

are probably better established than the annual concentration, the C_m value of 180 ug/m^3 and τ_b = 19.8 hrs might be the best parameters for the formulation of an air pollution index.

Figure 4 shows the BR curves for CO generated using the 1-hr and 8-hr standards set by two air control agencies, the Ministry of the Environment, Ontario, and the United States E.P.A. We see that while the 1-hr standard set by MOE is lower than that set by E.P.A., the 8-hr MOE concentration is higher than the E.P.A., standard. This difference implies that MOE standards are based on a biological response time of 1.79 hrs while the E.P.A. implicity assumes that the human body takes a longer time of 3.96 hours to recover from CO dosage. We also note that the MOE curve indicates that no adverse effects result from a continuous exposure of 12.8 ppm of CO. On the other hand, the E.P.A. curve fixes this long term concentration at a lower value of 7.81 ppm. It is necessary to recall that the preceding interpretation of the CO standards is based entirely on the hypothesized biological response model.

In the same figure we have drawn straight lines to indicate the consequences of a CO data line which passes through the points specified by the standards. We see that as the straight lines lie above the BR curves between the standard points, meeting the 1-hr and 8-hr standards does not ensure that the criteria set by the BR curve are met. In view of this, we feel that the best way of setting CO standards is by using the criteria derivation procedure described in section 3. In fact, for pollutants such as CO which are associated with relatively short biological response times, it would be necessary to use a dynamic model to stay below the specified injury level.

6. SUMMARY

A simple dynamic model of biological response to pollutant dosage can be used to derive a plausible form of the biological response curve. We have shown how the BR curve can be used to establish air quality criteria. We feel that the formalism of the derivation is important by itself. the BR curves. However, as the 3-hr. and 24-hr. concentration

The response model can also form the basis of an air pollution index which avoids the arbitrariness of currently available indices. The index is derived from sound physical principles and is thus preferable to other indices even if the parameters α and C_m have to be chosen in a less than satisfactory manner.

Although the BR curve "looks" like a straight line on a log-log graph (See Fig.3) for small αt, we cannot entirely reconcile our result with those of Larsen (1977). However, we note that the suggested BR curve reduces to the form given by O'Gara (Heck and Brandt, 1977) for plant response. This can be demonstrated as follows: For small αt, (3) can be written as

$$C_{at} = \frac{C_m}{[1-\exp(-\alpha t)]} \simeq \frac{C_m}{\alpha t\left(1-\frac{\alpha t}{2}\right)} \simeq \frac{C_m}{\alpha t}\left[1+\frac{\alpha t}{2}\right] \quad (9a)$$

or

$$C_{at} = \frac{C_m}{\alpha t} + \frac{C_m}{2} \quad (9b)$$

Equation (9b) is of the form given by O'Gara who obtained it by fitting a curve to his experimental data.

We realize that the determination of α and C_m for various pollutant-receptor pairs presents major difficulties. The importance of these parameters is tied to the validity of our model. However, even if the model is not realistic the principles behind the derivation of air quality criteria and our air pollution index have importance of their own.

REFERENCES

Heck, W.W., and Brandt, C.S., 1977: Effects on Vegetation. Air Pollution, Vol. 11, Third Edition, Arthur C. Stern, Ed., Academic Press, New York, 158-220.

Larsen, R.I., 1971: A Mathematical Model for Relating Air Quality Measurements to Air Quality Standards. Publ.AP-89, U.S. Environmental Protection Agency, Research Triangle Park, N.C. U.S.A.

Larsen, R.I., Gardner, D.E., and Coffin, D.L., 1977: An air quality data analysis system for interrelating effects, standards, and needed source reductions: Part 5. Nitrogen Dioxide Mortality in Mice. Paper presented 70th Annual Meeting of the Air Pollution Control Association, Toronto, Ontario, Canada. June 20 - 24, 1977.

McGuire, T., and Noll, K.E., 1971: Relationship between concentrations of atmospheric pollutants and averaging time. Atmospheric Environment, 5, 291-298.

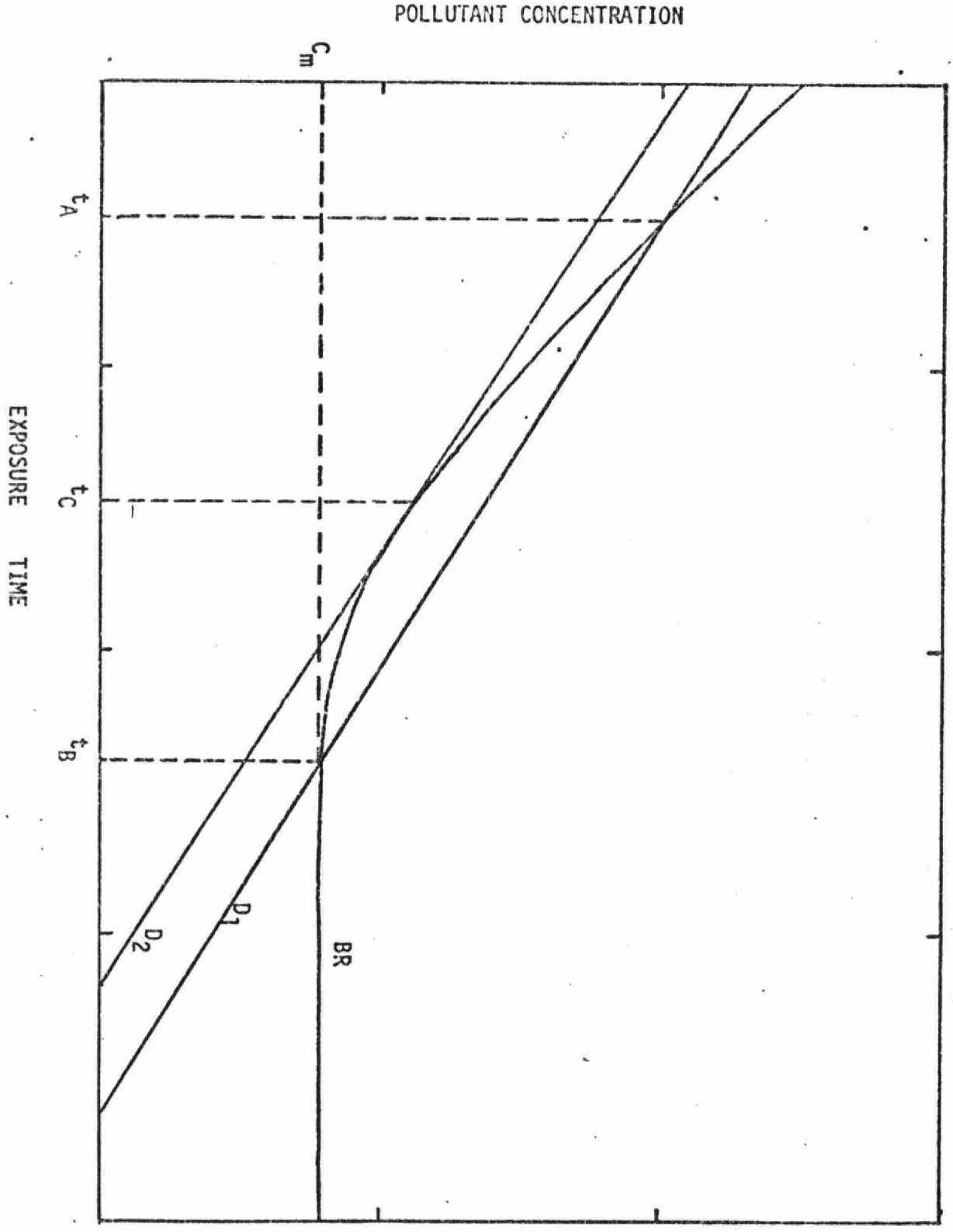
Munn, R.E., Phillips, M.L., and Sanderson, H.P.1977: Environment effects of air pollution implications for air quality criteria, air quality standards and emission standards. The Science of the Total Environment, 8, 53-67.

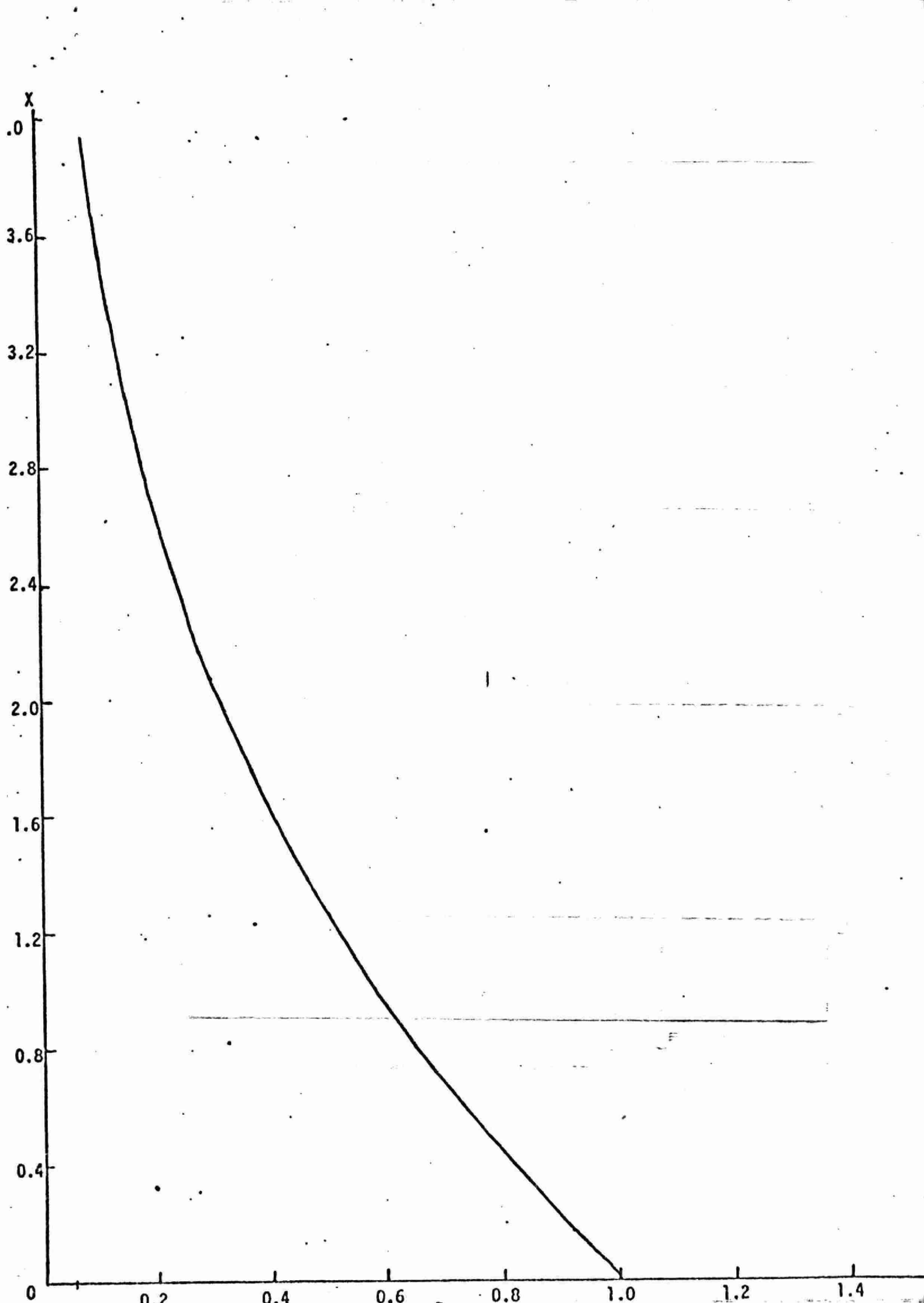
Roach, S.A., 1966: A more rational basis for air sampling programmes. Amer. Ind. Hyg. Assoc. J., 27, 1-12.

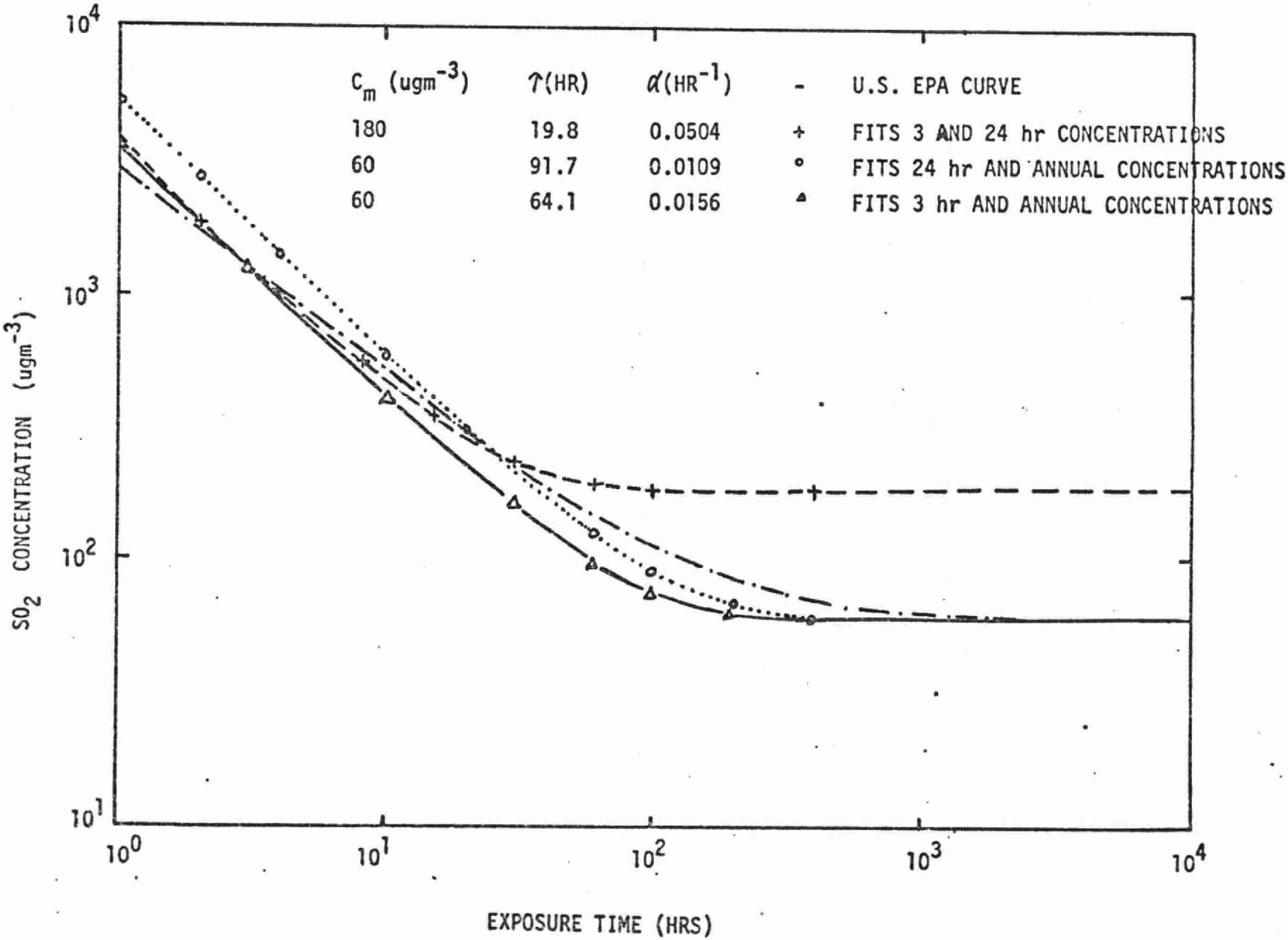
Thom. G.C., and Ott, W.R., 1975: Air Pollution Indices Sponsored by the Council on Environmental Quality and the Environmental Protection Agency, Available from U.S. Government Printing office, Washington, D.C. U.S.A.

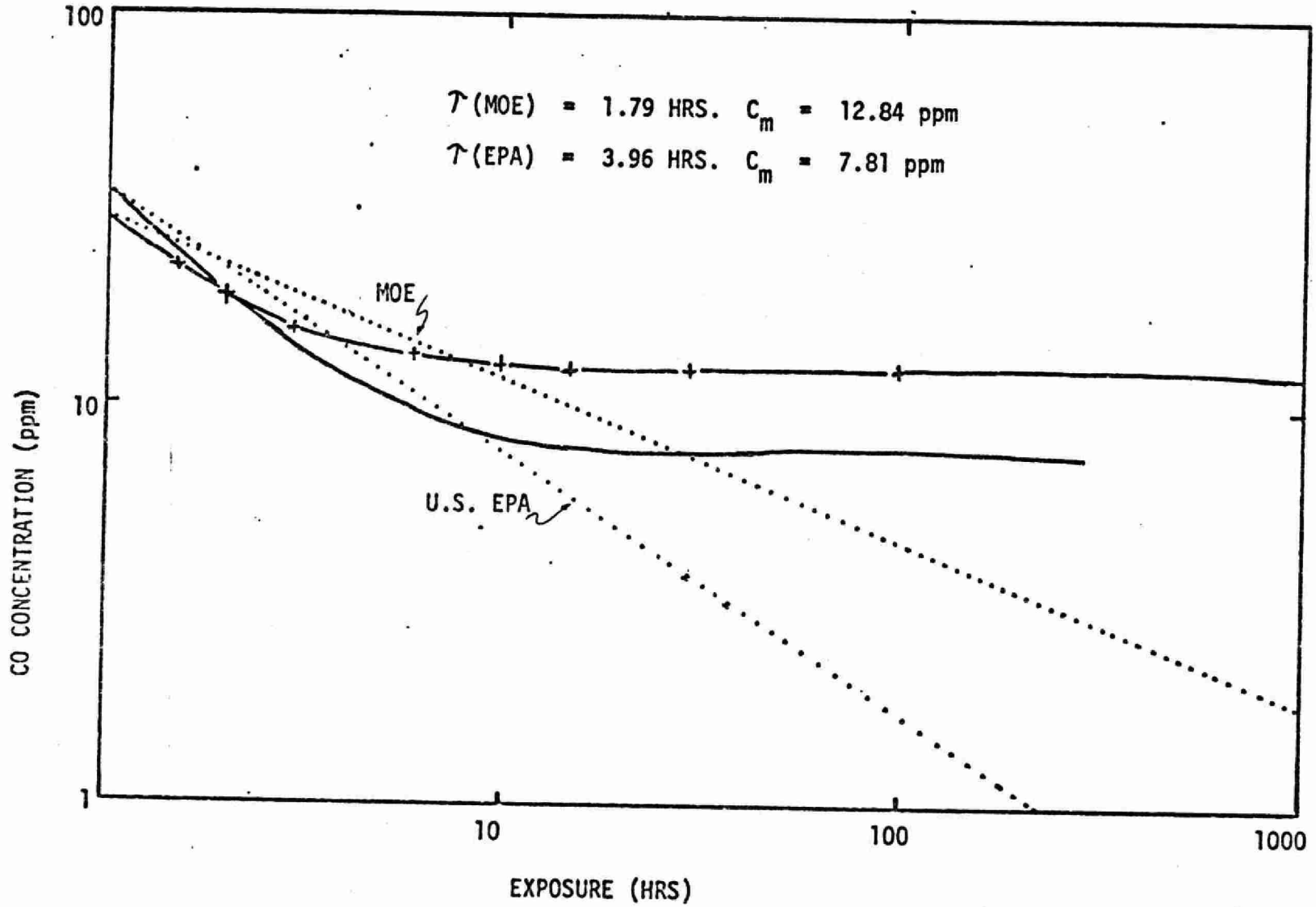
Saltzman, B.E., 1970: Significance of sampling time on air monitoring.J.Air Poll.Contr.Assoc., 20, 660-665.

LIST OF FIGURES


POLLUTANT CONCENTRATION
C_m
t_A
t_C
t_B
EXPOSURE TIME
D_2
D_1
BR


X
.0
3.6
3.2
2.8
2.4
2.0
1.6
1.2
0.8
0.4
0
0.2
0.4
0.6
0.8
1.0
1.2
1.4


C_m (ugm^{-3})
τ(HR)
α(HR^{-1})
180 19.8 0.0504
60 91.7 0.0109
60 64.1 0.0156
- U.S. EPA CURVE
+ FITS 3 AND 24 hr CONCENTRATIONS
° FITS 24 hr AND ANNUAL CONCENTRATIONS
Δ FITS 3 hr AND ANNUAL CONCENTRATIONS
SO_2 CONCENTRATION (ugm^{-3})
10^4
10^3
10^2
10^1
10^0
10^1
10^2
10^3
10^4
EXPOSURE TIME (HRS)


100
10
1
10
100
1000
τ(MOE) = 1.79 HRS. C_m = 12.84 ppm
τ(EPA) = 3.96 HRS. C_m = 7.81 ppm
MOE
U.S. EPA
CO CONCENTRATION (ppm)
EXPOSURE (HRS)

	DATE DUE		